Embolization of Dural Arteriovenous Malformation of the Jugular Bulb

- Case Report -

Jung Yong Ahn, M.D.,[1] Seong Oh Kwon, M.D.,[1]
Byung-Hee Lee, M.D.,[2] Eun Wan Choi, M.D.[2]

Departments of Neurosurgery,[1] Radiology,[2] Pundang CHA Hospital, Pochon CHA University, Sungnam, Korea

The authors report a 39-year-old male suffering pulsatile tinnitus of the left ear with dural arteriovenous malformation of the jugular bulb. There was a venous hypertension due to partial occlusion of the internal jugular vein and sigmoid sinus and venous drainage to the contralateral sinuses. Superselective embolization of these feeding arteries was successfully performed using 25% mixture of histoacryl and lipiodol. After embolization, his complaints of pulsatile tinnitus and buzzing noise behind his left ear disappeared. We present a case of dural arteriovenous malformation involving the jugular bulb, which completely disappeared after transarterial embolization with liquid adhesive.

KEY WORDS : Arteriovenous malformation · Embolization · Tinnitus.

Introduction

Dural arteriovenous malformations(dAVMs) consist of arteriovenous shunts of blood confined within dural leaflets and account for 10 - 15% of all intracranial arteriovenous malformations[8]. The most common location of dAVMs is in the transverse and sigmoid venous sinuses[1], with the nidus invariably localized at the transverse/sigmoid junction. But dAVM involving the jugular bulb is extremely rare. Symptoms and signs of the dural arteriovenous malformations can include pulse-synchronous tinnitus, bruit, headache, papilledema, hemorrhage, proptosis, visual decline, altered mental status, and transient or permanent neurological deficits[6,8].

The cause and pathogenesis of dAVMs remain unclear. A few cases reported in children suggest that these dAVMs are congenital, but dAVM has been generally thought to be an acquired lesion[2,6,9]. The association between dAVMs and sinus thrombosis has been well recognized[9], but a clear cause and effect has not been proven.

Direct surgical resection of dAVM has become an accepted treatment, but significant hemorrhage can result. Advances in microcatheter and guidewire technology have made superselective embolization of feeding arteries technically easier.

The authors report a case of dAVM of the jugular bulb, which had pulsatile tinnitus as the presenting symptom and was treated successfully with therapeutic embolization.

Case Report

A 39-year-old man presented with a 5-year-history of left ear tinnitus. There was no history of head trauma. A neurological examination at the time of admission revealed pulsatile bruit on the left retromastoid area. Magnetic resonance imaging(MRI) revealed an extraaxial mass that had isosignal intensity to gray matter on T2-weighted images(Fig. 1A) and had high signal intensity on T1-weighted images, which was homogeneously enhanced with gadolinium contrast media(Fig. 1B). Left external carotid angiography revealed an early visualization of sigmoid sinus and jugular vein by dAVM supplied especially from inferior tympanic artery and neuromeningeal branches of the ascending pharyngeal artery of the left external carotid artery and located on the jugular bulb(Fig. 2). The internal jugular vein and sigmoid sinus were partially occluded on venous phase angiogram and left venous drainage was diverted to the contralateral sinuses. From a transarterial approach, Prowler microcatheters(Cordis, FL, USA) were navigated into the above feeding arteries from the external carotid artery, which were then embolized with 1.8ml 25% mixture of histoacryl and lipiodol(Glue).

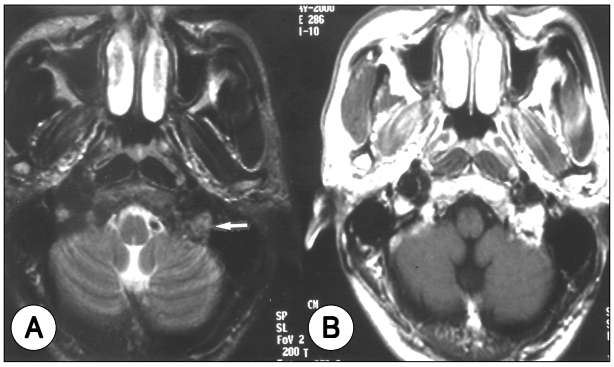

Fig. 1. Magnetic resonance imaging revealing an extraaxial mass(arrow) that has isosignal intensity to gray matter on T2-weighted image(A), which is homogeneously enhanced with gadolinium contrast media(B).

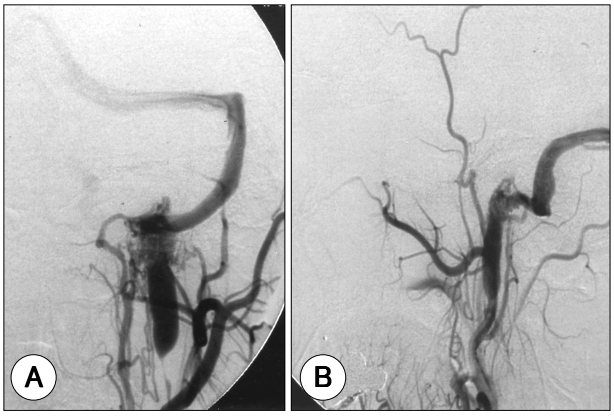

Fig. 2. Left external carotid angiograms(A : anteroposterior, B : lateral view) revealing an early visualization of sigmoid sinus and jugular vein by dural arteriovenous malformation supplied from inferior tympanic artery and neuromeningeal branches of the ascending pharyngeal artery of the left external carotid artery.

Follow-up external carotid angiogram demonstrated no dAVM and no visualization of the early venous drainage(Fig. 3). The patient was free from tinnitus after embolization and exhibited no neurological deficits.

Discussion

DAVMs are unique vascular abnormalities comprising numerous tiny connections between branches of dural arteries and a venous sinus with the nidus. Essential defining features are the nidus of the malformations and the early appearance of venous structures during the arterial phase of angiography[1]. The most common location of dAVMs is in the transverse and sigmoid venous sinuses[1], with the nidus invariably localized at the transverse/sigmoid junction. But dAVM involving the jugular bulb that is the internal jugular vein/sigmoid sinus junction area has not been previously documented. Its clinical presentation is similar with tumor of the jugular glomus. Harris et al[5]. reported a dAVM or a jugular glomus tumor as a possible cause of pulsatile tinnitus. The audible sound may result from turbulence in the blood stream, secondary to a change in the blood stream, and to a change in the blood's velocity as it passes through an arteriovenous fistula or a highly vascularized tumor of the jugular glomus. In this present case of a dAVM, the intraluminal stenosis itself might be responsible for the turbulence.

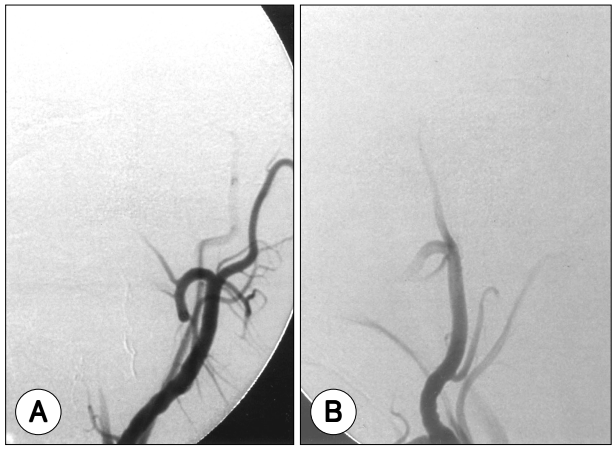

Fig. 3. Follow-up external carotid angiograms(A : anteroposterior, B : lateral view) demonstrating no dural arteriovenous malformation and no visualization of the early venous drainage.

The cause and pathogenesis of dAVMs are controversial, but are classified into two major categories, congenital and acquired. DAVMs have a very high incidence of abnormalities of venous channels. These abnormalities are described as irregularity and rigidity of the wall, stenosis and septation with filling defects in the lumen, and retrograde flow with partial or complete occlusion of the draining venous sinus or vein. Such changes are believed to be due to venous thrombosis, which is thought to be responsible for the occasional disappearance of these lesions[7]. Thrombosis or thrombophlebitis of the draining vein or sinus appears to be a common feature of these lesions and may be related to their origin[6]. Awad et al[1]. classified the progression of dAVMs into three stages. They speculated that sinus thrombosis and the opening of embryonic arteriovenous communication played an important role in the onset of this disease. This hypothesis was supported by sinus thrombosis with tumor[10], trauma, and craniotomy. Nishijima et al[9]. proposed that the mechanisms of the progression of dAVM's are as follows : 1) a single arteriovenous fistula forms in the dura near a venous sinus, 2) arterial blood begins to flow into the sinus via com-

municating dural veins, leading to formation of thrombosis, 3) pressure in the venous sinus increases, and this increase in internal pressure along with the organization of thrombi causes fibrous thickening of the sinus intima and interruption or proliferation of the elastic lamina of the sinus, 4) stenosis or occlusion of the sinus lumen is formed. In our case, there was no thrombosis of the sinus, but stenosis of the internal jugular vein and sigmoid sinus. Our case can be explained with Nishijima's hypothesis ; however, it does not prove that there is a cause and effect.

The theapeutic strategy for dAVMs includes transarterial embolization[3], transvenous embolization[4], and surgical resection of the involved sinus. Placement of embolic materials within the nidus of the malformation can result in cure. Halbach et al[3]. reported that only 59% of patients treated was completely cured by transarterial embolization. Although transvascular embolization of these pathways is technically possible, the risk of embolic reflux and stroke makes these less desirable. If embolic material flows through the nidus and occludes venous drainage, aggravation of symptoms may result, with diversion of venous drainage into cortical pathways. Although surgical excision of the involved sinus is possible, massive intraoperative hemorrhage can occur because of the rich vascularity of the surrounding structures. We used a transarterial approach with the placement of embolic materials, liquid adhesives, within the nidus. Because of the high arterial pressure within the involved sinus, there is often retrograde drainage of blood away from the sinus into cortical veins. Disconnection is important, if liquid adhesive embolic agents alone are contemplated, to prevent the flow of embolic agents into these cortical veins. When these connections have been interrupted and the sinus isolated by occluding its outflow, complete stasis is often observed after injection of contrast material or embolic agents. To prevent this potentially devastating complication and to eliminate the need for surgical interruption of draining veins, metal coils was often placed into the affected sinus with transvenous approach. Our case was not proper candidate for coil embolization with transvenous approach, because venous channels were already stenotic and venous hypertension was aggravated.

In conclusion, we report a case of dAVM involving the jugular bulb with partial occlusion of the internal jugular vein and sigmoid sinus. The dAVM completely disappeared after transarterial embolization with liquid adhesive.

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• **Address for reprints** : Jung Yong Ahn, M.D., Department of Neurosurgery, Pundang CHA Hospital, 351, Yatap-dong, Bundang-gu, Seongnam, 463-712, Korea
Tel : 031) 780-5262, Fax : 031) 780-5269
E-mail : jyahn@cha.ac.kr

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